CLAIMS:

1. A method for ameliorating the effects of a medical disorder such as a proliferative and/or inflammatory skin disorder in a mammal, said method comprising contacting the proliferating and/or inflamed skin or skin capable of proliferation and/or inflammation or a cell otherwise involved with said medical disorder with an effective amount of a nucleic acid molecule or chemical analogue thereof capable of inhibiting or otherwise reducing growth factor mediated cell proliferation and/or inflammation and/or other medical disorders.

2. A method according to claim 1 wherein cell proliferation and/or inflammation or other medical disorder is mediated by at least one of insulin-like growth factor I (IGF-I), keratinocyte growth factor (KGF), transforming growth factor-α (TGFα), tumour necrosis factor-α (TNFα), interleukin (IL) -1 (IL-1), IL-4, IL-6, IL-8 and/or basic fibroblast growth factor (bFGF).

3. A method according to claim 2 wherein cell proliferation and/or inflammation or other medical disorder is mediated by IGF-I.

4. A method according to claim 1 wherein the nucleic acid molecule inhibits or otherwise reduces IGF-I mediated cell proliferation and/or inflammation or other medical disorder.

5. A method according to claim 1 wherein the proliferative or inflammatory skin disorder is psoriasis, ichthyosis, pityriasis, rubra, pilaris, serborrhoea, keloids, keratosis, neoplasias, scleroderma, warts, benign growths or cancers of the skin.

6. A method according to claim 5 wherein the skin condition is psoriasis.

7. A method according to claim 1 wherein the other medical disorder is a hyperneovascular condition such as a neovascular condition of the retina, brain or skin, growth factor-mediated malignancies, other sclerotic disease, kidney disease or hyperproliferation of the inside of blood vessels or any other hyperplasia.

8. A method according to claim 1 wherein the mammal is a human.

9. A method according to claim 1 wherein the nucleic acid molecule is capable of inhibiting, reducing or otherwise interfering with IGF-I-interaction with its receptor.

10. A method according to claim 9 wherein the nucleic acid molecule is an antisense molecule capable of reducing expression of a gene encoding IGF-I, IGF-I-receptor or an IGF binding protein (IGFBP).

11. A method according to claim 10 wherein the nucleic acid molecule is an antisense molecule capable of reducing expression of a gene encoding IGFBP-2, -3, -4, -5 or -6.

12. A method according to claim 11 wherein the nucleic acid molecule is an antisense molecule capable of reducing expression of a gene encoding IGFBP-2 or IGFBP-3.

13. A method according to claim 10 wherein the antisense molecule is at least about 15 nucleotides in length and is capable of interacting with at least one sequence selected from the list set forth in Example 6 or Example 7 or Example 8.

14. A method according to claim 12 wherein the antisense molecule comprises the nucleotide sequence:

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5'-ATCTCTCCGCTTCCTTTC-3' (<400>10)

15. A method according to claim 12 wherein the antisense molecule is selected from the following:

UCCGCAGCCAGACUU (<400>12)
CACAGUUGCUGCAAG (<400>13)
UCUCCGCUUCCUUUC (<400>14)
AGCCCCCACAGCGAG (<400>15)
GCCUUGGAGAUGAGC (<400>16)
UAACAGAGGUCAGCA (<400>17)
GGAUCAGGGACCAGU (<400>18)
CGGCAAGCUACACAG (<400>19)
GGCAGGCAGGCACAC (<400>20)

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16. A method according to claim 15 wherein the antisense molecule is <400>12, <400>13 or <400>14.

17. A method according to claim 15 wherein the antisense molecule is <400>12.

18. A nucleic acid molecule comprising at least about 10 nucleotides capable of hybridising to or forming a heteroduplex or otherwise interacting with a complementary form of <400>12 to <400>20 inclusive.

19. A nucleic acid molecule comprising at least about 15 nucleotides capable of hybridising to or form a heteroduplex or otherwise interacting with a complementary form of <400>12 to <400>14 inclusive.

20. A method of ameliorating the effects of psoriasis or other medical disorder, said method comprising contacting proliferating skin or skin capable of proliferation or cell otherwise associated with said medical disorder with an effective amount of one

or more nucleic acid molecules or chemical analogues thereof capable of inhibiting or otherwise reducing IGF-I mediated cell proliferation other medical disorder wherein said one or more molecules comprises a polynucleotide capable of interacting with mRNA directed from an IGF-I gene, an IGF-I receptor gene or a gene encoding an IGFBP.

21. A method according to claim 20 wherein the IGFBP is IGFBP-2 or IGFBP-3.

22. A method according to claim 20 wherein the mammal is a human.

23. A method according to claim 22 wherein the nucleic acid molecule is capable of interacting with a nucleotide sequence selected from the list set forth in <400>12 to <400>14 inclusive.

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24. A method according to claim 23 wherein the nucleic acid molecule comprises the nucleotide sequence selected from <400>12 to <400>14.

25. A composition comprising a nucleic acid molecule capable of inhibiting or otherwise reducing IGF-I mediated cell proliferation or other medical disorder said composition further comprising one or more pharmaceutically acceptable carriers and/or diluents.

26. A composition according to claim 25 wherein the nucleic acid molecule is antisense molecule to a gene encoding IGF-I, IGF-I-receptor or an IGFBP.

27. A composition according to claim 26 wherein the nucleic acid molecule is selected from <400>12 to <400>20 inclusive.

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28. A composition according to claim 26 selected from <400>12 to <400>14 inclusive.

29. A method for ameliorating the effects of a proliferative and/or inflammatory skin disorder such as psoriasis said method comprising contacting the proliferating and/or inflamed skin or skin capable of proliferation and/or inflammation with effective amounts of UV treatment and a nucleic acid molecule or chemical analogue thereof capable of inhibiting or otherwise reducing IGF-I mediated cell proliferation and/or inflammation.

30. A method according to claim 29 wherein the proliferative or inflammatory skin disorder is psoriasis, ichthyosis, pityriasis, rubra, pilaris, serborrhoea, keloids, keratosis, neoplasias, scleroderma, warts, benign growths or cancers of the skin.

31. A method according to claim 30 wherein the proliferative or inflammatory skin disorder is psoriasis.

32. A method according to claim 29 wherein the nucleic acid molecule is capable of inhibiting, reducing or otherwise interfering with IGF-I-interaction with its receptor.

33. A method according to claim 32 wherein the nucleic acid molecule is an antisense molecule capable of reducing expression of a gene encoding IGF-I, IGF-I-receptor or an IGF binding protein (IGFBP).

34. A method according to claim 33 wherein the nucleic acid molecule is an antisense molecule capable of reducing expression of a gene encoding IGFBP-2, -3, -4, -5 or -6.

35. A method according to claim 34 wherein the nucleic acid molecule is an antisense molecule capable of reducing expression of a gene encoding IGFBP-2 or IGFBP-3.

36. A method according to claim 33 wherein the nucleic acid molecule is an antisense molecule capable of reducing expression of a gene encoding IGF-I receptor.

37. A method according to claim 29 wherein the antisense molecule is at least about 15 nucleotides in length and is capable of interacting with at least one sequence selected from the list set forth in Example 6 or Example 7 or Example 8.

38. A method according to claim 37 wherein the antisense molecule comprises the nucleotide sequence:

5′-ATCTCTCCGCTTCCTTTC-3′ (<400>10)

39. A method according to claim 37 wherein the antisense molecule is selected from the following:

UCCGGAGCCAGACUU (<400>12)
CACAGUUGCUGCAAG (<400>13)
UCUCCGCUUCCUUUC (<400>14)
AGCCCCCACAGCGAG (<400>15)
GCCUUGGAGAUGAGC (<400>16)
UAACAGAGGUCAGCA (<400>17)
GGAUCAGGGACCAGU (<400>18)
CGGCAAGCUACACAG (<400>19)
GGCAGGCAGGCACAC (<400>20)

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40. A method according to claim 39 wherein the antisense molecule in <400>12, <400>13 or <400>14.

41. A method according to claim 40 wherein the antisense molecule in <400>12.

42. A method according to claim 39 wherein the UV treatment occurs simultaneously with or following contact with the nucleic acid molecule or its chemical analogue.

43. Use of an antisense molecule directed to the gene encoding IGF-I receptor or its mRNA as adjunct therapy in combination with UV treatment to reduce proliferation and/or inflammation of keratinocyte cells.

44. Use according to claim 43 in the treatment of psoriasis.

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